REMARKS

Claims 29-31 are in this application. It is respectfully requested that the amendment of claims 29 to 31 to cancel the phrase "preventing or" be entered.

There is information in this application and known to those of skill in the art that complications of diabetes which is caused by insulin resistance or impaired glucose tolerance can be prevented. There is data in the application which establishes that compounds of this invention reduce blood glucose levels, lower cholesterol and lowers triglycerides. By reducing blood glucose levels, it is possible to prevent and/or treat diabetes and prevent and/or treat complications of diabetes.

There are references to support that the compounds of the present invention have beneficial effects. These references also support that the compounds can be useful in the mitigation or prevention of the complications of diabetes. The following literature references suggest that insulin resistance and hyperinsulineamia is associated with other complications such as high blood pressure, microvascular angina, coronary heart disease, atherosclerotic coronary artery disease, chronic kidney disease and cardiovascular disease (Ref: Baillieres Clin Endocrinol Metab 1993 Oct;7&4):1063-78); Diabetes 1988 Dec;37(12):1595-607; Am J Hypertens 1989 Jun;2(6 Pt);419-23; J Cardiol 1998 Nov; 32(5):291-300; Metabolism 1992 May;41(5 supp; 1);16-9; Diabete Metab 1991 May;17(1 Pt2):78-86; J Am Soc Nephrol 2003 Feb;14(2):469-77; Journal of clinical endocrinology and metabolism Vol;83, No:8, 2773-2776; MMW Fortschr Med. 2000, June 22, 142 (25);42-4).

The following reference suggests, the prevention of hyperglycemia and insulin resistance by PPAR gamma agonist in animal models (Diabetes Vol 50, Oct-2001, 2316-2322;

American Physiological society 1996, E742-E747). The reference, Kidney Int 2001 May; 59(5):1899-910 demonstrates *in vivo*, that the PPAR gamma ligand TGL ameliorates the progression of glomerulosclerosis in an nondiabetic model.

The following references describe the use of PPAR agonists for the prevention and/or treatment of *in vivo* are available for specific diseases or conditions

1) Use of PPAR agonists for the pathogenesis of conditions associated with Syndrome X. (Life Sci. 2000, Oct. 6,67(20):2405-16; Ann, N Y Acad. Sci. 2002, 967:28-33)

2) Use of PPAR agonists against cancer (Mol. Cancer Ther., 2002 Mar, 1 (5):357-63)

3) Use of PPAR agonists against pancreatitis (Pancreas, 2002 Mar, 24 (2): 184-90)

4) Use of PPAR agonists against inflammatory bowel diseases (Gasteroenterol Clin. Biol., 2000, 24 (8-9): 719-24)

5) Use of PPAR agonists in the pathogenesis of Alzhemer's disease (CNS Drugs 2003, 17 (1): 27-45)

6) Use of PPAR agonists against cognitive functions in dementia. (Curr. Med. Chem., 2002, Jan., 9(1),83-8).

7) Use of PPAR agonists against psoriasis. (Arch. Dermatol, 2000, May, 136(5),609-16

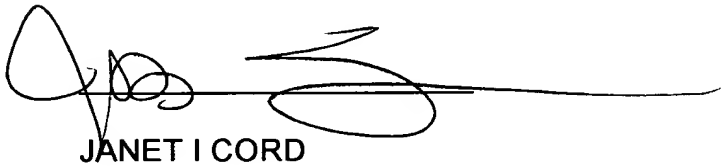
8) Use of PPAR agonists against endothelial cell activation. (Biochem. Biophys. Res. Commun., 2002, May, 293(5), 1431-37

9) Use of PPAR agonists against retinopathy. (Invest. Ophthalmol. Vis. Sci., 2000, 41(8), 2308-17; Arch. Ophthalmol., 2001, 119(5), 709-17)

10) Use of PPAR agonists against osteoporasis (Nippon Rinsho, 2000, 58(2), 456-60; Br, J. Oharmacol., 2000, 130(3), 495-504))

Therefore, based on the above it is clear that claims 29-31 are enabled.

Respectfully submitted,



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